IN THE CLAIMS:

Following listing of claims is submitted to replace the listing of claims in this application.

Please cancel claims 1-17 and 28-40.

Please add the new claims 41-50.

1-17 CANCELLED.

18. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual to an in vivo pharmaceutical comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein by detecting the genetic modification in the nucleic acid comprising SEQ ID NO: 2, wherein the genetic modification is a substitution of cytosine by thymidine at position 825 ~~and/or at position 1429~~ of SEQ ID NO:2, and wherein the thymidine at position 825 of SEQ ID NO: 2 is indicative of the individual having increased activation capacity of G proteins which is indicative of the reduced responsiveness of the individual to the in vivo pharmaceutical.

19. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual to in vivo ~~to~~ hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which stimulate the G protein subunit GalphaS comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein, wherein the genetic modification is a substitution of cytosine by thymidine at position 825 ~~and/or at position 1429~~ of SEQ ID NO:2, wherein the thymidine at position 825 of SEQ ID NO: 2 is indicative of reduced responsiveness of the individual to in vivo hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G

protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which stimulate the G protein subunit GalphaS.

20. (PREVIOUSLY PRESENTED) The method of claim 18 or 19, further comprising determining the presence of the Arg16Gly variant and the Gln27Glu variant in the beta2 adrenergic receptor.

21. (PREVIOUSLY PRESENTED) The method of claim 18, wherein pharmaceutical is erythropoietin.

22. (PREVIOUSLY PRESENTED) The method of claim 18, wherein the pharmaceutical is an immunosuppressive and the development of hypertension during such therapy is evaluated.

23. (PREVIOUSLY PRESENTED) The method of claim 22, wherein the immunosuppressive is cyclosporin.

24. (PREVIOUSLY PRESENTED) The method of claims 19 or 20, wherein the pharmaceutical is for treatment and prevention of a migraine headache.

25. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual to treatment with beta-adrenoceptor blockers comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymidine position 825 ~~and/or position 1429~~ of SEQ ID NO:2, wherein the presence of thymidine at position 825 of SEQ ID NO: 2 is indicative of the individual having intensified reduction of the cardiac output as a response to treatment with beta-adrenoceptor blockers.

26. (CURRENTLY AMENDED) A method for evaluating responsiveness of an individual in treatment with a substance having prostoglandin E1 action comprising evaluating the individual for a genetic modification in a gene enclosing a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymidine position 825 ~~and/or position 1429~~ of SEQ ID NO:2, wherein the presence of thymidine at position 825 of SEQ ID NO: 2 is indicative of the individual being less responsive to the substance having prostaglandin E1 action.

27. (PREVIOUSLY PRESENTED) The method of claim 26, wherein the substance is prostaglandin E1.

Claims 28-40 CANCELLED.

41. (NEW) A method for evaluating responsiveness of an individual to an in vivo pharmaceutical comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein by detecting the genetic modification in the nucleic acid comprising SEQ ID NO: 2 or a polypeptide encoded by a nucleic acid comprising the SEQ ID NO:2, wherein the genetic modification is a substitution of cytosine by thymidine at position 1429 of SEQ ID NO:2, and wherein the thymidine at position 1429 of SEQ ID NO: 2 is indicative of the individual having increased activation capacity of G proteins which is indicative of the reduced responsiveness of the individual to the in vivo pharmaceutical.

42. (NEW) A method for evaluating responsiveness of an individual to in vivo to hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which

stimulate the G protein subunit GalphaS comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein, wherein the genetic modification is a substitution of cytosine by thymine at position 1429 of SEQ ID NO:2, wherein the thymidine at position at position 1429 of SEQ ID NO: 2 is indicative of increased decreased responsiveness to in vivo to hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which stimulate the G protein subunit Galpha .

43. (NEW) The method of claim 41 or 42, further comprising determining the presence of the Arg16Gly variant and the Gln27Glu variant in the beta2 adrenergic receptor.

44. (NEW) The method of claim 41, wherein pharmaceutical is erythropoietin.

45. (NEW) The method of claim 41, wherein the pharmaceutical is an immunosuppressive and the development of hypertension during such therapy is evaluated.

46. (NEW) The method of claim 45, wherein the immunosuppressive is cyclosporin.

47. (NEW) The method of claims 42 or 43, wherein the pharmaceutical is for treatment and prevention of a migraine headache.

48. (NEW) A method for evaluating responsiveness of an individual to treatment with beta-adrenoceptor blockers comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymine position 1429 of SEQ ID NO:2, wherein the presence of thymidine at position 825 of SEQ ID NO: 2 is indicative of the

individual having intensified reduction of the cardiac output as a response to treatment with beta-adrenoceptor blockers.

49. (NEW) A method for evaluating responsiveness of an individual in treatment with a substance having prostoglandin E1 action comprising evaluating the individual for a genetic modification in a gene enclosing a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymine position 1429 of SEQ ID NO:2, wherein the presence of thymidine at position 825 of SEQ ID NO: 2 is indicative of the individual being less responsive to the substance having prostaglandin E1 action.

50. (NEW) The method of claim 26, wherein the substance is prostaglandin E1.